THE JOURNAL OF INVESTIGATIVE DERMATOLOGY, 82:386–388, 1984

Vol. 82, No. 4
Printed in U.S.A.

LETTERS TO THE EDITOR

We are pleased to receive Letters to the Editor on appropriate subjects. These letters should be submitted in typewritten form, double-spaced, and are not to exceed 2½ pages. When appropriate, we will solicit comments from the original authors. All Letters to the Editor are subject to editing and possible abridgment.

AGE-RELATED CHANGES IN THE CUTANEOUS BASAL LAMINA

To the Editor:

Hull and Warfel [1] reported recently on age-related changes in basal lamina that were observed by SEM. They cited two papers [2,3] as providing "incomplete, inaccurate" interpretations of basal lamina structure. Unfortunately, Hull and Warfel did not understand the papers to which they referred. These papers deal with *fetal* skin and adult skin. The surface we describe in fetal skin as covered by microvilli is the periderm. "Pseudopodia" were not mentioned in either of these papers. The periderm is the *outermost* cell layer of fetal skin that is present up to 6 months gestational age. This is completely unrelated to the basal cell undersurface described by Hull and Warfel. Our only reference to the dermal-epidermal junction (DEJ) was in terms of TEM observations of lamina lucida, lamina densa, hemidesmosomes, anchoring filaments and anchoring fibrils in fetal skin; no observations of the contour of the DEJ were reported.

We think it is incumbent upon authors to read and understand the papers to which they refer, especially when the reference takes the form of unwarranted criticism.

REFERENCES

1. Hull MT, Warfel KA: Age-related changes in the cutaneous basal lamina: scanning electron microscopic study. J Invest Dermatol 81:378–380, 1983
2. Holbrook KA, Odland GF: The fine structure of developing human epidermis: light, scanning and transmission electron microscopy of the periderm. J Invest Dermatol 65:16–38, 1975
3. Holbrook KA, Smith LT: Ultrastructural aspects of human skin during embryonic, premature, neonatal, and adult periods of life. Birth Defects 17:9–38, 1981

Karen A. Holbrook, Ph.D.
George F. Odland, M.D.
Lynne T. Smith, M.A.
University of Washington
School of Medicine
Seattle, Washington

REPLY

Dr. Holbrook and her associates have taken umbrage unnecessarily. They misinterpreted our intent in citing their papers and our statements regarding the appearance of the DEJ. We referred to their works as fine examples of published TEMs of the DEJ that have appeared in the recent literature accessible to most of your readers. At no point do we attack the quality of their work regarding the periderm. We agree that their observations on the periderm are irrelevant to our work. Rather, we mean to convey that basing concepts on two-dimensional TEMs similar to those they published has led to the interpretation that the DEJ is characterized by pseudopodia and microvilli (not to be confused with the microvilli of the periderm). Although the authors insist that "no observations of the contour of the DEJ were reported" in their works, they have extensively illustrated the irregular contour of the DEJ, as it appears in a total of eighteen illustrations in both papers. Based on these, we believe that their two papers illustrate the DEJ about as often and as well as any others we've encountered in the recent literature.

We sense that their letter was written in anger and haste. The emotional and inflammatory tone of it is unwarranted. Of course, we read and understood their papers. And, of course, we understand the "concept" of periderm. We believe that their implications to the contrary are irresponsible.

Meredith T. Hull, M.D.
K. A. Warfel, M.D.
Indiana University School of Medicine
Indianapolis, Indiana

NEUTROPHILS IN PSORIASIS

To the Editor:

In a very interesting article by Fraki and associates published in the September 1983 issue [1] the authors referred to our findings on neutral serine proteinase activities in peripheral blood polymorphonuclear leukocytes (PMNLs) in psoriasis [2], and we believe that their comments are due to some misunderstanding.

We have found increased activities of neutral proteinases of PMNLs, not in all patients with psoriasis studied, as has been suggested [1], but only in patients with active plaque psoriatic lesions spreading peripherally [2]. In contrast to that, the activities of neutral proteinases—elastase and cathepsin G, as well as lysozyme—were markedly decreased in both patients with active guttate psoriasis and patients with stationary lesions [3]. Thus, when the mean activities of these enzymes were calculated for the total psoriasis group, no differences were established between psoriatics and normal controls, which is in agreement with the data of Fraki et al [1].

Regarding their remark concerning the method of extraction of granule enzymes [1], other authors also applied low ionic strength to solubilize these enzymes [4,5]. Rindler-Ludwig et al [6] suggested extraction of cathepsin G either by high ionic strength or by pH below 5.0, or both. Cohn and Hirsch [7] presented data on the lysis of isolated granules, which were destroyed by 0.004 M citric acid. We have used either one extraction with 0.05 M citrate buffer at pH 3.2 [2] or four sequential washings in 0.05 M citrate buffer at pH 3.5 (two of them with the addition of Triton X-100) [3]. The comparison of several extraction methods of neutral proteinases of PMNLs has been done in our laboratory, and the paper is in preparation.

We think that the discrepancy between our results [2,3] and those of Fraki et al [1] could be, to a certain extent, related to the sampling of the patients. It is conspicuous that about 50% (or more) of the patients studied by these authors had psoriasis plus arthritis, whereas cases of psoriatic arthritis were not included in our material.

Our studies indicate that the activities of neutral proteinases of PMNLs undergo the dynamic changes in the course of the disease, being normal or reduced shortly after reappearance of guttate lesions, increased during peripheral spreading of plaques, and again markedly decreased when the disease becomes stationary. Increased amounts of neutral proteinases released from PMNLs chemoattracted to the psoriatic epidermis might be of importance in the self-perpetuation of the disease.

REFERENCES

1. Fraki JE, Jakoi L, Davies AO, Lefkowitz RJ, Snyderman R, Lazarus GS: Polymorphonuclear leukocyte function in psoriasis: chemotaxis, chemokinesis, beta-adrenergic receptors, and proteolytic enzymes of polymorphonuclear leukocytes in the peripheral blood from psoriatic patients. J Invest Dermatol 81:254–257, 1983
2. Glinski W, Zarebska Z, Jablonska S, Imiela J, Nosarzewski J: The activity of polymorphonuclear leukocyte neutral proteinases and their inhibitors in patients with psoriasis treated with continuous peritoneal dialysis. J Invest Dermatol 75:481–487, 1980
3. Glinski W, Barszcz D, Zarebska Z, Janczura E, Jablonska S: Neutral proteases and other granule enzymes of neutrophils as related to activity, extent and duration of psoriasis (abstr). J Invest Dermatol 80:362, 1983
4. Olsson I, Odeberg H, Weiss J, Elsbach P: Bactericidal cationic proteins of human granulocytes, Neutral Proteinases of Human Polymorphonuclear Leukocytes. Edited by K Havemann, A Janoff. Baltimore/Munich, Urban and Schwartzenberg, 1978, pp 21–32
5. Ohlsson K: Purification and properties of granulocyte collagenase and elastase, Neutral Proteinases of Human Polymorphonuclear Leukocytes. Edited by K Havemann, A Janoff. Baltimore/Munich, Urban and Schwarzenberg, 1978, pp 89–101
6. Rindler-Ludwig R, Bretz U, Baggioline M: Cathepsin G: the chymotrypsin-like enzyme of polymorphonuclear leukocytes, Neutral Proteinases of Human Polymorphonuclear Leukocytes. Edited by K Havemann, A Janoff. Baltimore/Munich, Urban and Schwarzenberg, 1978, pp 138–149